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**A further demonstration of the part played by accessory food factors in the ætiology of rickets.** By EDWARD MELLANBY.

In a previous demonstration to this Society of puppies showing the part played by diet in producing and preventing Rickets, it was seen that, using a standard rachitic diet of bread and milk (175 c.c.), the disease was prevented by butter, cod liver oil and most other fats tested, increased milk (500 c.c.), meat and meat extracts, but was not prevented by yeast, linseed oil and the proteins of milk and meat. On the basis of these results, an attempt was made to improve the standard rachitic diet in order to produce a more rapid development of the disease and to render it less easy to prevent. In the dogs demonstrated today, the standard diet has been separated milk (175–350 c.c.), 70 per cent. wheaten bread *ad lib.*, linseed oil 5–10 c.c., yeast 5–10 grams, orange or lemon juice 3 c.c., sodium chloride 2 grams. This diet will produce a rapid development of rickets, easily recognisable by X-ray photograph within 6 weeks of beginning the diet. In the puppies exhibited it will be seen that

(1) lean meat (10 grams a day) only delays but does not prevent rickets and that the extent of the delay depends on the initial weight and rate of growth of the animal. The greater the rate of growth, the less is the effect of meat.

(2) increasing the separated milk to 350–400 c.c. per diem does not prevent rickets. This excludes the possibility that the calcium intake is deficient.

(3) 10 grams of butter or cod liver oil, even on the above diet, completely prevents rickets.

(4) 10 grams of cotton seed oil, olive oil or babassu oil per diem does not prevent rickets when substituted for linseed oil.

All vegetable oils are not equally ineffective in preventing rickets. Linseed oil is the worst of those tested, then in order of increasing merit, tentatively suggested, babassu, cotton-seed, peanut (*arachis*) and olive. A hydrogenated fat tested was similar to linseed oil. Suet and lard appear (experiment not completed) to prevent rickets if 10 grams of meat per diem is also eaten. A small amount of meat helps an otherwise ineffective fat.

The above facts are for the most part in keeping with the idea that rickets is a disease primarily due to a deficiency of Fat-soluble A. Animal fats are the best preventatives.

It is difficult, however, to fit the inhibitory action of lean meat, meat and malt extracts into this theory.

Other facts brought out in this work are:

(1) the more rapidly growing the dog, the more anti-rachitic factor is required to keep the growth straight.

(2) while not yet able to deny the necessity of Fat-soluble A being present in a diet to ensure growth, it is the case that many of my more rapidly growing puppies have had the least Fat-soluble A. This accessory factor seems to be more essential for ensuring normal growth as opposed to abnormal growth and keeping the tissues in good working order.

(3) taking this work in conjunction with my wife's research on factors affecting the formation of teeth<sup>1</sup>, it appears that those substances containing and associated with Fat-soluble A are particularly concerned in the calcification processes of bones and teeth. Further, the close relationship between rickets and defective teeth (both as regards enamel formation and arrangement in the jaws) is placed on an experimental basis.

<sup>1</sup> The effect of diet on teeth formation. May Mellanby, *Lancet*, December 7, 1918.

